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COOPERATIVE STOCHASTIC EFFECTS IN GLOBALLY COUPLED INTERACTING NEURONS; STOCHASTIC RESONANCE

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ABSTRACT

We consider a network of N nonlinearly coupled neuron-like elements subject to Langevin noise and a weak periodic modulation. Through the adiabatic elimination proceedure, the dynamics of a single neuron is obtained from the coupled stochastic differential equations describing the network. The bifurcation properties of this "reduced neuron" model are discussed, together with cooperative stochastic effects (e.g. stochastic resonance) that result from the presence of the modulation.

Recently, there has been an upsurge of interest in *single* or few-neuron nonlinear dynamics [1-4]. However, the precise relationship between the many-neuron connected model and a single effective neuron dynamics has not been examined in detail. Such a reduced neuron dynamics has been derived recently [5] for a network of N symmetrically interconnected neuron-like elements embodied, for example in the "connectionist" models of Hopfield [6,7] or Shamma [8] (the latter corresponding to a mammalian auditory network). Through an adiabatic elimination procedure, the dynamics (in closed form) of a single neuron has been obtained from the system of coupled differential equations describing the N-neuron problem. The problem has been treated both deterministically and stochastically (through the inclusion of additive and multiplicative noise terms). It is important to point out that the work of ref [5] does not include *a priori* a self-coupling term; rather, that theory results in an explicit form of the self-coupling term, in terms of the parameters of the remaining neurons in the network i.e., the network dynamics is reduced to a closed equation describing a single neural element with nonlinearity determined by the parameters describing the remaining neurons in the network. This "reduced" or "effective" neuron model is expected to reproduce some of the gross features of biological neurons. Multiplicative noise effects in the model have been discussed, in greater detail, in [9]. The fact that simple single neuron models, such as the model to be considered in this work, can indeed reproduce several features observed in biological experiments has been demonstrated [10] through the construction of inter-spike-interval histograms (ISIHs) using a Schmidt trigger to model the neuron. The results of this simple model are in very good qualitative agreement with data obtained in two different experiments, on two different animals.

In this work, we extend the work of [5] to include, *a priori*, self-coupling terms (such terms are almost always present in electronic neural networks, for example) as well as a weak low-frequency periodic modulation. Thus, we consider a general network of neuron-like elements described by the dynamics,

$$C_i \dot{u}_i = \sum_{j=1}^{N} J_{ij} \tanh u_j - \frac{u_i}{R_i} + F_i(t) + q \sin \omega t . \tag{1}$$

Here, u_i represents the membrane potential of the ith neuron with input capacitance C_i and a leakage current due to the trans-membrane resistance R_i. $F_i(t)$ is taken to be Gaussian delta-correlated noise having zero mean and variance σ_i^2 (we assume that the noise sources for the different neurons are uncorrelated, throughout this work) and the dot denotes time-differentiation. It is important to note that an equation of the form (1) describes, in general, N nonlinearly coupled bistable oscillators. This model is also the continuum version of the Hopfield model [7] if the coupling coefficients J_{ij} are computed using the Hebb rule. We do not assume such a rule for computing the coefficients J_{ij}, nor do we assume that the matrix J is symmetric. Our system (1) therefore is closer to neurophysiological models than the Hopfield model. We now assume that at least two distinct timescales exist in the dynamics described by (1). Specifically we assume that

$$R_i \ll R_1 \quad (i > 1). \tag{2}$$

This is tantamount to assuming that we may treat the neuron described by the membrane potential u_1 as a "slow" neuron interacting with a "bath" of $N-1$ "fast" neurons that are in equilibrium. We may then adiabatically eliminate the bath variables from (1) and write down an equation for the variable u_1 in terms of the bath variables. The proceedure for doing this has been described in [5] and is not repeated here. Specifically, an N-body Fokker Planck equation is constructed from (1). Using Haken's slaving principle [11] the N-body probability density function is factored into a density function $g(u_1)$ describing the "slow" variable u_1 and a conditional density function embodying the remaining variables. This permits the separation of the Fokker Planck equation into fast and slow components. The Fokker Planck equation for the bath variables is integrated, in the steady state, by invoking a local equilibrium assumption (this is necessary since detailed balance is usually not satisfied in two or more dimensions). Ultimately, we arrive at a Fokker Planck equation for u_1 whence a stochastic differential equation for the variable u_1 (this variable now characterizes the "effective" or "reduced" neuron) may be written down by inspection. This equation is,

$$\dot{u}_1 = -\alpha u_1 + \beta \tanh u_1 + \delta \sin \omega t + \sqrt{\sigma_e^2} F(t), \tag{3}$$

where,

$$\alpha = (R_1 C_1)^{-1}; \quad \beta = C_1^{-1} \left[J_{11} + G^{-1} \sum_{i \neq 1} R_i J_{1i} J_{i1} \left(1 - \frac{\sigma_i^2 R_i}{2 C_i} \right) \right];$$

$$\delta = \frac{q}{C_1} \left[1 + G^{-1} \sum_{i \neq 1} R_i J_{1i} \left(1 - \frac{\sigma_i^2 R_i}{2 C_i} \right) \right]; \quad G \equiv 1 - \sum_{i \neq 1} J_{ii} R_i; \quad \sigma_e^2 \cong \sigma_1^2 / C_1. \tag{4}$$

In carrying out the proceedure leading to (3) we have assumed further that the modulation frequency is smaller than the Kramers frequency of the unmodulated system. This "adiabatic assumption" allows us to treat the modulation as being approximately constant in our solution of the Fokker Planck equation. This assumption is also a cornerstone of the adiabatic theory of stochastic resonance [12] on which our subsequent results are based. Further, we assume that

$$\sigma_i^2 R_i \ll 2 C_i \quad (i \neq 1). \tag{5}$$

This assumption (quantified in [5]) guarantees the convergence of the steepest descent techniques used to evaluate the coefficient β in (3). Note also the absence of terms involving coupling between pairs of "fast" neurons; these terms are $O(R_i R_j)$ $(i,j > 1)$ or higher and can be neglected. It is worth pointing out that the adiabatic elimination proceedure, based on the inequality (2) may actually have some basis in neurophysiology, e.g., when one considers the interaction of a single neuron with a "dendritic bath".

A detailed analysis of the full dynamics described by (3) is beyond the scope of this Letter and will be presented elsewhere. In the following, therefore, we focus our attention on a single "slow" neuron u_1 coupled to a "fast" neuron u_2 as a representative example which we use to describe the cooperative effects that arise as a result of their interaction. Since neurons are (inherently) bistable elements, it is instructive to study the bifurcation properties of this system which, in the absence of the noise and modulation terms, may be described by the potential function,

$$U(u_1) = \frac{\alpha}{2} u_1^2 - \beta \ln \cosh u_1. \tag{6}$$

For positive α and β, the potential is bimodal if $\beta/\alpha \geq 1$ with the minima located (for large β/α) at $c \approx \pm \frac{\beta}{\alpha} \tanh \frac{\beta}{\alpha}$ and an elliptic point at $u_1 = 0$. It is instructive to consider the effects of the fast neuron dynamics on the transition to bimodality in the potential. To this end, we plot, in figure one, the ratio β/α as a function of the resistance R_2, for different values of the variance σ_2^2 of the noise source in the fast neuron. In this figure, we take $J_{11} = 1$ and we have chosen α to guarantee that, in the absence of the second neuron ($J_{12} = 0$), the potential corresponding to the neuron u_1 is bimodal. Since the ratio β/α also determines the barrier height (for a given α), one observes that dramatically different effects are obtained depending on the sign of the product $J_{12}J_{21}$ in (4). These effects are also readily observable in equations (4). For $J_{12} = 1 = J_{21}$, the coupling to the "fast" neuron is seen to increase the ratio β/α upto a maximum value, occurring at $R_2 = \frac{1 - (1 - 2C_2 J_{22}/\sigma_2^2)^{1/2}}{J_{22}}$, after which it decreases (note that the quantity inside the square root must be positive in order to get an extremum within the constraints inherent in the inequality (5)); the opposite effect occurs in the $J_{21} = -1$ case. As the noise σ_2^2 increases, the deviation from the $J_{12} = 0$ case becomes less pronounced. At $R_2 = 0$ and $2C_2/\sigma_2^2$, the curves for $J_{21} = 1$ and $J_{21} = -1$ intersect, yielding the $J_{12} = 0$ result ($\beta/\alpha = J_{11}R_1$). Beyond this intersection, the inequality (5) is violated and we do not expect the theory to yield accurate results.

We now consider the effects of the deterministic modulation. Specifically, we are interested in the phenomenon of stochastic resonance wherein a small amount of noise can introduce correlated switching events in the effective potential (6). An adiabatic theory, valid for very low frequency ω and weak amplitude q (such that there is no switching in the absence of noise) has been developed by McNamara and Wiesenfeld [12]. The central result of this theory is that if one computes the signal-to-noise-ratio (SNR) of a nonlinear system of the form (3) as a function of the noise variance, then the SNR passes through a maximum at a noise variance approximately equal to the potential barrier height. Stochastic resonance has been investigated [13] for a single (isolated) oscillator of the form (3), with arbitrary α and β (see also the recent review [14]). In the current context, we define the deterministic switching threshold as the critical value δ_c of the scaled modulation amplitude δ, above which one would obtain deterministic switching in the $\sigma_1^2 = 0 = \sigma_2^2$ case. This critical value can be easily found to be given by $\delta_c = -\alpha u_c + \bar\beta \tanh u_c$ where $u_c \equiv \ln \left[\sqrt{\frac{\beta}{\alpha}} + \sqrt{\frac{\beta}{\alpha} - 1} \right]$ and $\bar\beta \equiv (\beta)_{\sigma_2^2 = 0}$. Then, in order to satisfactorily explain the stochastic resonance using adiabatic theory we must ensure that $\delta < \delta_c$ and $\omega < \omega_k$, the Kramers rate for the unmodulated system (the latter condition is also necessary in the derivation of (3)). Figure two shows the characteristic signal-to-noise-ratio (SNR) curve (obtained via numerical integration of (3)) as a function of σ_1^2. The curve is peaked at $\sigma_c^2 = U_0$, the barrier height determined from (6). The effect of introducing noise of varying amounts in the fast neuron dynamics is also elucidated in this figure. In figure three we plot the peak SNR (computed from the the adiabatic theory [12,13] for $\sigma_c^2 = U_0$), normalized to its value in the $J_{12} = 0$ case (no coupling to the bath neuron). We consider the $J_{12} = 1 = J_{21}$ case. It is evident that the efficiency of the stochastic resonance process is enhanced (for

low values of σ_i^2) by the coupling to the bath neuron, with the most pronounced improvement occurring for low noise strengths in the fast neuron. Similar effects have been observed recently in a mean field model of globally coupled bistable oscillators [15]. The increase in the SNR with R_2 (keeping R_1 fixed) is seen to correlate with the decrease of the ratio β/α (and the accompanying decrease in the potential barier height) observed in figure one for $J_{21} = -1$. In fact one may show, within the framework of the adiabatic theory [12,13] and for large β/α, that an enhancement of the peak SNR such as observed in figure 2 (and in figure 3 below) is obtained if $\dfrac{\delta^2}{q^2}\sqrt{\dfrac{\beta}{J_{11}}} > 1$, for a given α. Note however, that increasing σ_2^2 tends to decrease β/α as well as the effective interaction δ. This explains the degradation of the effect as σ_2^2 increases. The corresponding effect for the $J_{21} = 1$ case is very small; this is readily understood by observing that the potential barrier height increases for this case, as R_2 is increased from zero. Hence, an enhancement in the SNR (as observed in figure 2 for the $J_{21}=-1$ case) does not occur within the range of values of R_2 that is allowed by (2) and (5). In passing, we briefly point out that the case $J_{12} = -1 = -J_{21}$ is qualitatively similar to the $J_{12} = 1 = -J_{21}$ case, insofar as the magnitude of the parameter β is concerned; however, the effective signal amplitude δ is seen to be lowered in this case (compared to the $J_{12}=1$ case) so that the signal-to-noise ratio is also degraded. Similar arguments apply to the $J_{12} = -1 = J_{21}$ case.

The analysis of the preceeding paragraph underscores the importance of the *sign* of the interaction. This is also obvious from the definitions (3) wherein we observe that β (and hence the potential barrier height) depends on the product $J_{ij}J_{ji}$ whereas the effective modulation term δ depends only on J_{1i} (the factor G^{-1} also plays a role but we do not consider its effect in this Letter). Clearly, in order to achieve the best possible SNR the barrier height should be decreased and the effective modulation increased (but not beyond the threshold for deterministic switching) as a result of the coupling to the bath neurons; this is best achieved by having (for the two-neuron case under consideration here) $J_{12} > 0, J_{21} < 0$. In neurophysiological terms, we could argue that having a distribution of excitory and inhibitory interactions provides superior performance to having all the couplings of one type (the latter situation is also unlikely to occur in neurophysiology, although it could certainly be realized in electronic analogs).

Finally, we consider the case $J_{11} = 0$. For this case, the potential in the absence of any coupling ($J_{12}=0$) is parabolic. However, as apparent in figure four, the coupling to the fast neuron can render the effective potential (6) bimodal above a certain critical value of R_2. This critical value is given by $R_{2c} \equiv \dfrac{C_2}{J_{12}J_{21}R_1\sigma_2^2}\left[(J_{12}J_{21}R_1 + J_{22})^2 - 2J_{12}J_{21}R_1\sigma_2^2\right]^{1/2}$. Unlike the preceeding case, however, this bimodality (which actually corresponds to a bifurcation in the most probable value of u_1 [9,16]) only occurs for $J_{12}J_{21} > 0$ within the constraints imposed by (5). Further, large noise strengths σ_2^2 tend to degrade this effect. Nonetheless, it is apparent that one will observe a stochastic resonance effect in the presence of the coupling to the fast neurons even though there is no such effect in the isolated ($J_{12}=0$) case.

The above work may be extended to larger networks. Clearly, as evidenced by the structure of the factor G^{-1} in (4) as well as the nature of the terms occurring within the summations, the magnitudes as well as the *signs* of the coefficients J_{ij} are of paramount importance in determining how the network performs. Note that, for a general network of nonlinear oscillators the coefficients J_{ij} could take on any values, as long as the system remains stable i.e., $\beta \geq 0$ (if $0 \leq \beta < \alpha$, the potential (6) will be parabolic). However, to maintain some contact with neural network theory, we have assumed $|J_{ij}| < 1$ in all our simulations. The calculations reported here have been extended to larger networks of the form (1) in which the resistances R_i and the variances $\sigma_i^2(i > 1)$ are drawn from a uniform random set subject to the condition (2) and (5). The results obtained are qualitatively similar to those discussed here for the special case $i = 2$, and will be presented in an upcoming publication. It is tantalizing to speculate that *neurophysiological systems constantly adjust*

the synaptic couplings J_{ij} (which may depend, in general, on a vast array of internal parameters) *in response to the stimulus and noise, such that the potential function that characterizes the network* (for our model, this would be the "reduced" or "effective" potential (6)) *admits of more than one minimum.* This leads, in turn, to nonlinear dynamic effects such as stochastic resonance, in which the noise actually enhances the flow of information throught the system; this effect appears to improve as a result of the connections to the "bath" neurons. As pointed out earlier, adjusting the internal parameters such that $\beta/\alpha \geq 1$ also guarantees stability; in the neighbourhood of the minima of (6) one may write [17] the long-time probability density function for u_i in the form $\exp[S]$ where S is a generalized "entropy" taking the form, $S = -\frac{1}{2} \frac{d^2 U}{du_i^2}(c) \delta u_i^2$. S is a Liapounov function in the neighbourhood of the potential minima. If the work of this Letter could be generalized to the point of solving the Fokker Planck equation corresponding to the fully coupled system (1) in the steady state, the potential function would be an N-dimensional hypersurface. To date there does not exist a technique for deriving such a potential for an N-dimensional nonlinear stochastic system.

From the standpoint of this Letter it is also important to reiterate the beneficial effects of coupling the neuron u_1 to the "fast" neuron u_2. The coupling clearly enhances the information flow (measured by the SNR) to the output of the system and can, for not too large noise variances σ_i^2 provide signal processing capabilities that are outside the capabilities of the original oscillator ($J_{ij}=0$). The addition of noise to the deterministic dynamics significantly enhances the signal processing characteristics of the network. This was pointed out earlier by Buhmann and Schulten [18,19] who stated that *noise...is an essential feature of the information processing capabilities of the neural network and not a mere source of disturbance better suppressed..."*

Acknowledgements

Bibliography

1. Z. Li and J. Hopfield; Biol. Cyber. 61, 379 (1988).
2. H. Tuckwell; "Stochastic Processes in the Neurosciences," (SIAM, Philadelphia 1988).
3. M. Paulus, S. Gass and A. Mandell; Physica D40, 135 (1990).
A144, 333 (1990).
5. W. Schieve, A. Bulsara and G. Davis; Phys. Rev. A43, 2613 (1990)
6. J. Hopfield; Proc. Nat. Acad. Sci. 79, 2554 (1982).
7. J. Hopfield; Proc. Nat. Acad. Sci. 81, 3088 (1984).
8. S. Shamma; in "Methods in Neuronal Modelling," eds. C. Koch and I. Segev (MIT Press, Cambridge, MA 1989).
9. A. Bulsara and W. Schieve; Phys. Rev. A44, 7913 (1991).
10. A. Longtin, A. Bulsara and F. Moss; Phys. Rev. Lett. 67, 656 (1991).
11. H. Haken; "Synergetics" (Springer Verlag, Berlin, 1977).
12. B. McNamara and K. Wiesenfeld; Phys. Rev. A39, 4854 (1989).
13. A. Bulsara, E. Jacobs, T. Zhou, F. Moss and L. Kiss; J. Theor. Biol. 154, 531 (1991).
14. F. Moss; Stochastic Resonance: From the Ice Ages to the Monkey's Ear; in "Some Problems in Statistical Physics" ed. G. Weiss (SIAM, Philadelphia 1992)
15. P. Jung, U. Behn, E. Pantazelou and F. Moss; Collective Response in Globally Coupled Bistable Oscillators; preprint.
16. W. Horsthemke and R. Lefever; "Noise-Induced Transitions", (Springer Verlag, Berlin 1984).

17. L. Landau and E. Lifshitz; "Statistical Mechanics" (Pergamon, 1969).

18. J. Buhmann and K. Schulten; in "Neural Networks for Computing" ed. J. Denker (AIP conference proceedings vol 151, 1986).

19. J. Buhmann and K. Schulten; Biol. Cyber. 56, 313 (1987).

Figure Captions

1. Effective nonlinearity parameter β/α (computed from (4) for the two-neuron case) vs R_2. $(R_1, J_{11}, J_{22}) \equiv (10.0, 1.0, 1.0)$. Solid curves represent $J_{12} = 1.0 = J_{21}$ with $\sigma_2^2 = 0, 10.0, 20.0$ reading from the top curve downward. Dotted curves correspond to $Jsub \, 12 = 1.0 = -J_{21}$ with $\sigma_2^2 = 0, 10.0, 20.0$ reading from the bottom curve upward.

2. Signal-to-noise ratio vs slow neuron noise variance σ_e^2. Bottom curve: $J_{12} = 0$ (isolated case); top curve: $J_{12} = 1.0 = -J_{21}$, $R_2 = 0.1, \sigma_2^2 = 0$; middle curve: $J_{12} = 1.0 = -J_{21}$, $R_2 = 0.1, \sigma_2^2 = 5.0$. $q = 0.1 = \omega$; frequency resolution $= 0.001$. Other parameters as in fig. 1.

3. Peak signal-to-noise-ratio vs. R_2 for $J_{12} = 1.0 = -J_{21}$ and $\sigma_2^2 = 0, 5.0, 10.0, 20.0$ reading from the top curve doownward. Other parameters as in fig. 2.

4. Effective nonlinearity parameter β/α vs R_2. $(R_1, J_{11}, J_{22}) \equiv (10.0, 0.0, 1.0)$. Solid curves represent $J_{12} = 1.0 = J_{21}$ with $\sigma_2^2 = 0, 5.0, 10.0$ reading from the top curve downward. Dotted curves correspond to $Jsub \, 12 = 1.0 = -J_{21}$ with $\sigma_2^2 = 0, 5.0, 10.0$ reading from the bottom curve upward.
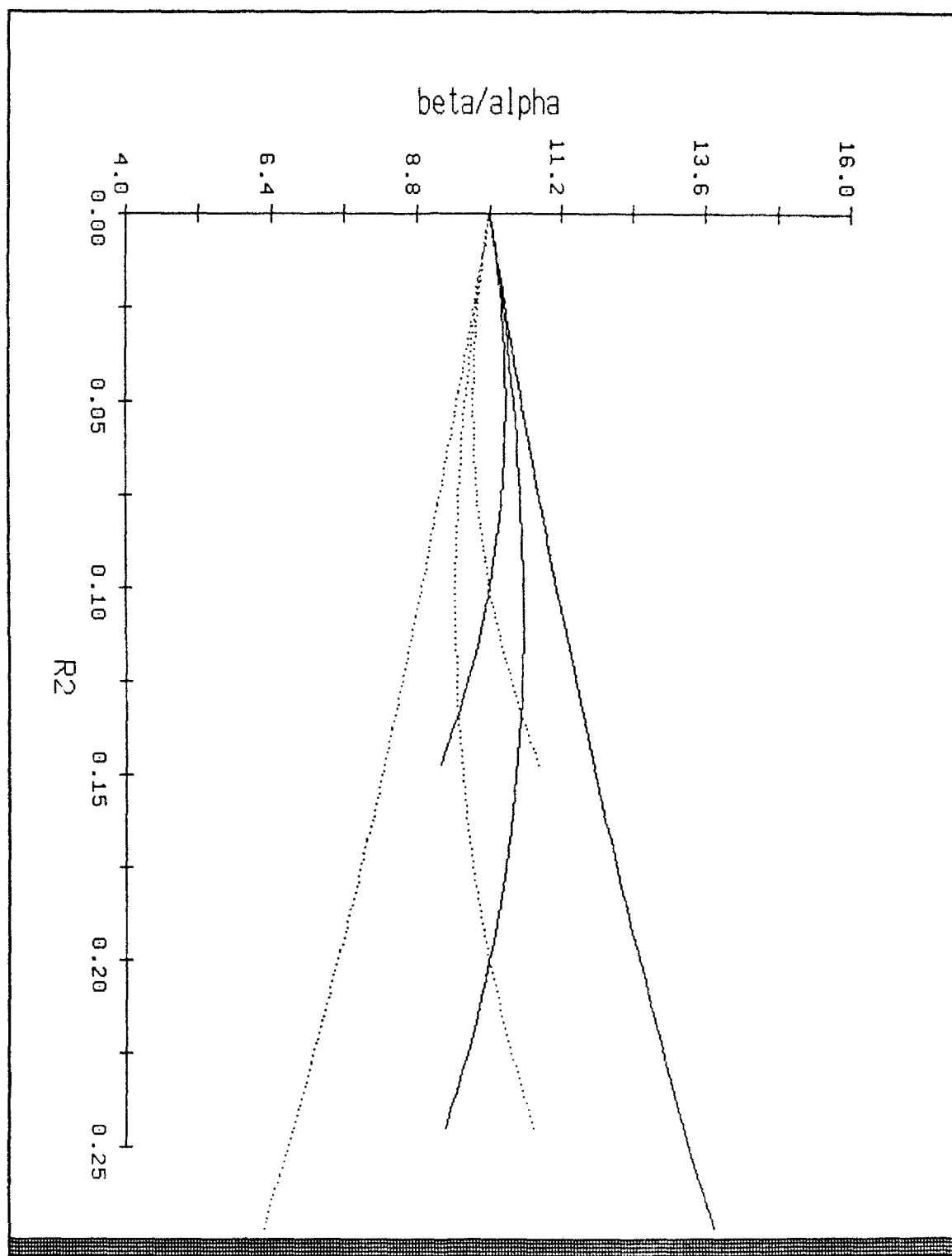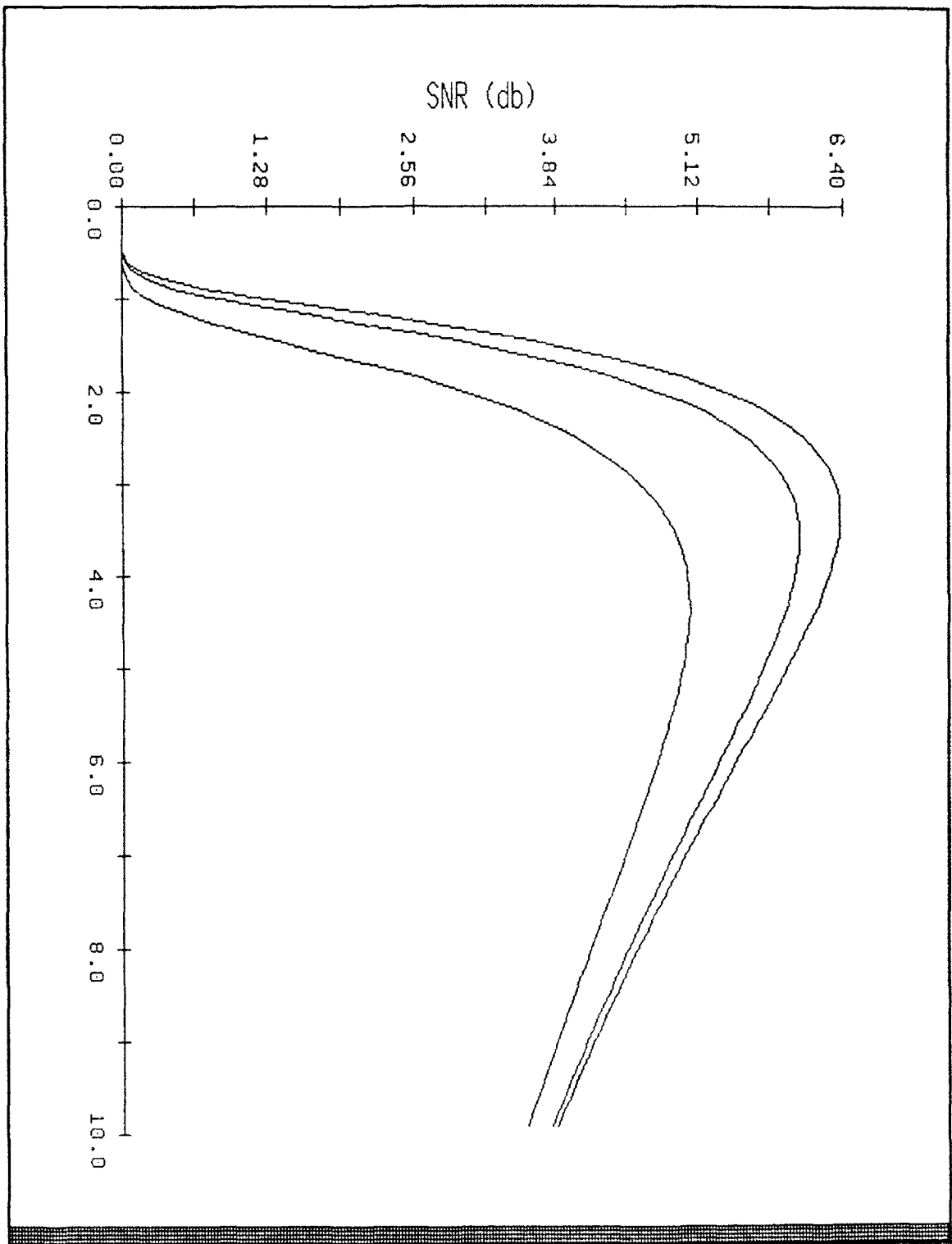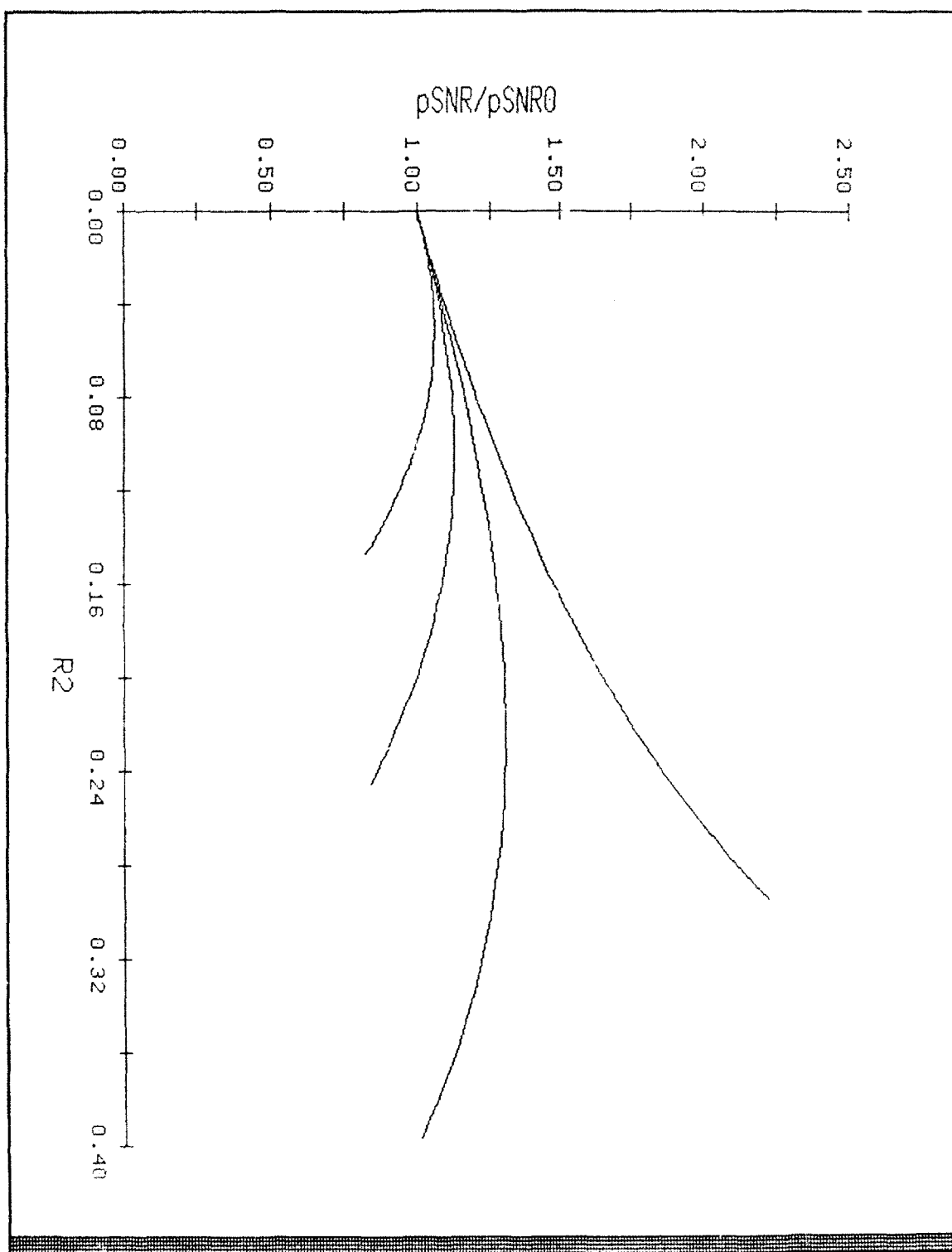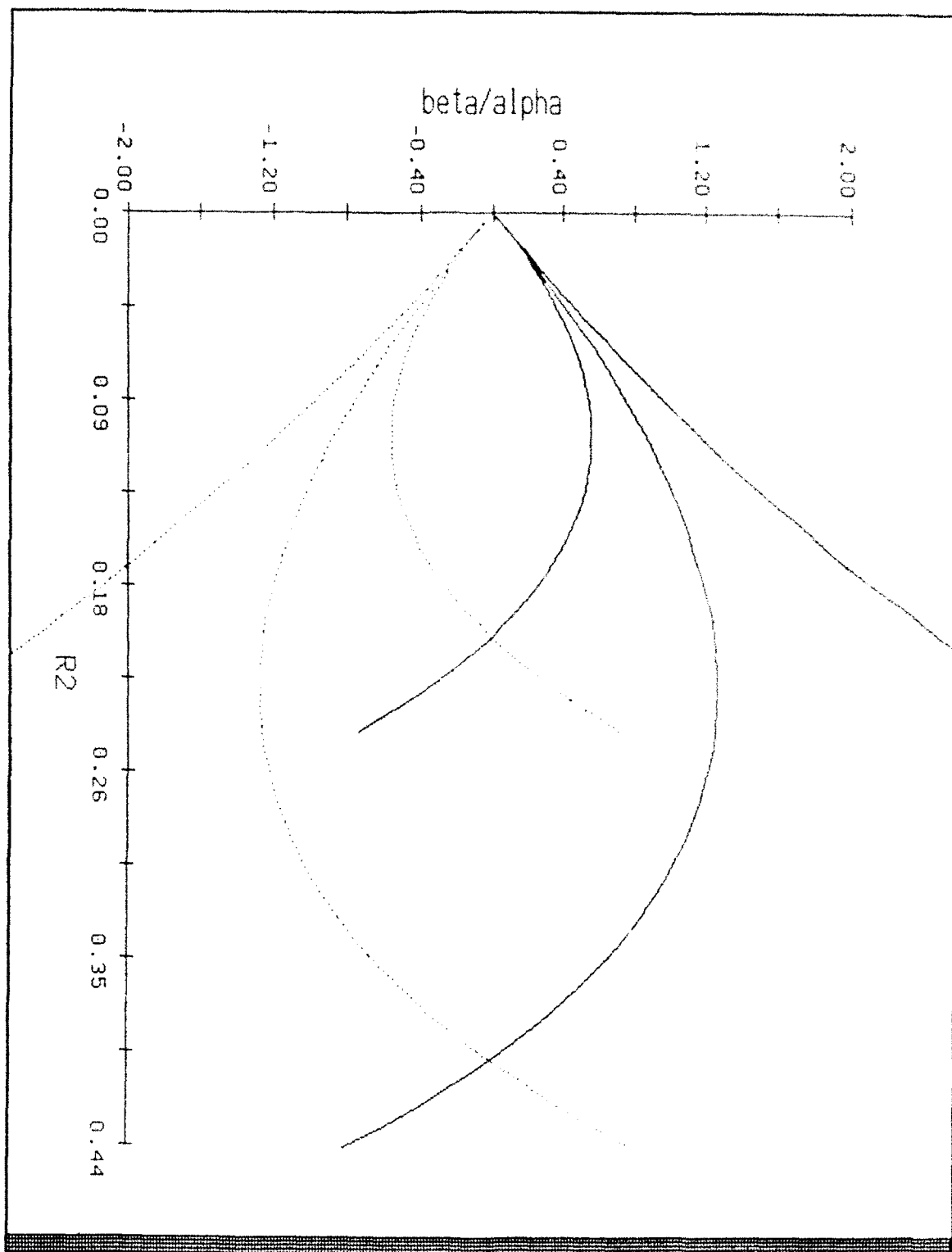
Fig 1

Fig 2

Fig 3

beta/alpha

R2

Fig 4